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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/584,653	12/03/2008	Torben Falk Orntoft	Sorge-653	8207
93599	7590	10/13/2011		
Eric P. Mirabel, JD, LL.M. 3783 Darcus Street Houston, TX 77005				
EXAMINER AEDER, SEANE				
ART UNIT 1642		PAPER NUMBER		
NOTIFICATION DATE 10/13/2011		DELIVERY MODE ELECTRONIC		

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

emirabel@comcast.net

Office Action Summary**Application No.**

10/584,653

Applicant(s)

ORNOT ET AL.

Examiner

SEAN AEDER

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 02 August 2011.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ An election was made by the applicant in response to a restriction requirement set forth during the interview on ____; the restriction requirement and election have been incorporated into this action.
- 4) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 5) ☒ Claim(s) 68, 70, 72-77, 79-81, 83-107, 121-123 and 128 is/are pending in the application.
- 5a) Of the above claim(s) 91, 121-123 and 128 is/are withdrawn from consideration.
- 6) ☐ Claim(s) ____ is/are allowed.
- 7) ☒ Claim(s) 68, 70, 72-77, 79-81, 83-90, and 92-107 is/are rejected.
- 8) ☐ Claim(s) ____ is/are objected to.
- 9) ☐ Claim(s) ____ are subject to restriction and/or election requirement.

Application Papers

- 10) ☐ The specification is objected to by the Examiner.
- 11) ☐ The drawing(s) filed on ____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 12) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. ____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftperson's Patent Drawing Review (PTO-942)
- 3) ☐ Information Disclosure Statement(s) (PTO/SB-08)
Paper No(s)/Mail Date ____
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date ____
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: ____

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Detailed Action

The Amendments and Remarks filed 8/2/11 in response to the Office Action of 5/5/11 are acknowledged and have been entered.

Claims 68, 70, 72-77, 79-81, 83-107, 121-123, and 128 are pending.

Claims 91, 121-123, and 128 remain withdrawn.

Claims 68, 70, 72-77, 79, 81, 84, 89, 92, 94-98, 102-107, 121, 122, and 128 have been amended by Applicant.

Claims 68, 70, 72-77, 79-81, 83-90, and 92-107 are currently under examination.

No species have been rejoined.

The following Office Action contains NEW GROUNDS of rejections necessitated by amendments.

Objections Withdrawn

All previous objections are withdrawn.

Rejections Withdrawn

All previous rejections are withdrawn.

New Rejections Necessitated by Amendments

Claim Rejections - 35 USC § 101

35 U.S.C. 101 reads as follows:

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Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

Claims 68, 70, 72-77, 79-81, 83-90, and 92-96 are rejected under 35 U.S.C. 101 because claims 68, 70, 72-77, 79-81, 83-90, and 92-96 encompass methods that merely involve abstract mental processes. Claims 68, 70, 75-77, 79-81, 83-90, and 92-96 can be performed by merely looking at printed results and mentally "determining" and "classifying". Abstract mental processes are not patentable. See Benson, 409 U.S. at 67 ("Phenomena of nature, ...mental process, and abstract intellectual concepts are not patentable, as they are the basic tools of scientific and technical work."). It is noted that instant claims 72-74 recite methods wherein gene expression products "are analyzed"; however, analyzing can be completed mentally and the claims do not recite active method steps of *detecting* expression of genes in samples.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 68, 70, 72-77, 79-81, 83-90, 92, 93, and 95-107 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for (1) methods wherein colon cancer patients with lower levels of MLH1 polynucleotides in colorectal tumor samples, as compared to the level of MLH1 polynucleotides in colorectal tumor samples from patients with hereditary

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disease, are classified as having a sporadic cancer, (2) methods wherein colon cancer patients with lower levels of PIWIL1 polynucleotides in colorectal tumor samples, as compared to the level of MLH1 polynucleotides in colorectal tumor samples from patients with sporadic disease, are classified as having a hereditary cancer, and (3) methods wherein colon cancer patients with lower levels of Hypothetical protein FLJ13842 polynucleotides in colorectal tumors, as compared to the level of Hypothetical protein FLJ13842 polynucleotides in colorectal tumor sample from patients with microsatellite stable disease, are classified as having microsatellite instable tumors (pages 67-69, in particular), **the specification does not reasonably provide enablement for** methods wherein a colon cancer patient with just any amount of just any polypeptide or polynucleotide expressed by a gene in Table 13 in just any sample from the colon cancer patient is classified, using just any determination(s) to classify, as having a hereditary cancer, a sporadic cancer, a microsatellite stable cancer, and a microsatellite instable cancer. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to practice the invention commensurate in scope with these claims.

Factors to be considered in determining whether undue experimentation is required are summarized in *Ex parte* Forman, 230 USPQ 546 (BPAI 1986). They include the nature of the invention, the state of the prior art, the relative skill of those in the art, the amount of direction or guidance disclosed in the specification, the presence or absence of working examples, the predictability or unpredictability of the art, the breadth of the claims, and the quantity of

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experimentation which would be required in order to practice the invention as claimed.

The instant claims are broadly drawn to methods wherein a colon cancer patient with just any amount of just any polypeptide or polynucleotide expressed by a gene in Table 13 in just any sample from the colon cancer patient is classified, using just any determination(s) to classify, as having a hereditary cancer, a sporadic cancer, a microsatellite stable cancer, and a microsatellite instable cancer. This includes methods wherein gene expression products are either polynucleotides or polypeptides. This further includes contradictory methods wherein, for example, elevated expression of a particular marker equally indicates both sporadic and hereditary cancer.

This invention is in a class of invention which the CAFC has characterized as "the unpredictable arts such as chemistry and biology". *Mycogen Plant Sci., Inc. v. Monsanto Co.*, 243 F.3d 1316, 1330 (Fed. Cir. 2001).

The specification teaches methods wherein a particular microsatellite status of a colorectal cancer tumor is determined based on higher or lower levels of particular polynucleotides disclosed in Table 17 and methods wherein downregulation of MLH1 polynucleotides in colorectal tumor samples is indicative of patients with sporadic disease and downregulation of PIWIL1 polynucleotides in colorectal tumor samples is indicative of patients with hereditary cases (pages 67-69, in particular). Such teachings demonstrate that the method does not function as claimed because downregulation of PIWIL1 polynucleotides in colorectal tumor samples is not indicative of patients with sporadic disease, as

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encompassed by the claims. The specification further teaches methods wherein colon cancer patients with lower levels of Hypothetical protein FLJ13842 polynucleotides in colorectal tumors, as compared to the level of Hypothetical protein FLJ13842 polynucleotides in colorectal tumor sample from patients with microsatellite stable disease, are classified as having microsatellite unstable tumors. Such a teaching demonstrates that the method does not function as broadly claimed because downregulation of Hypothetical protein FLJ13842 polynucleotides is not indicative of microsatellite stable tumors, as encompassed by the claims.

Undue experimentation would be required to determine how expression products other than MLH1 polynucleotides and PIWIL1 polypeptides would be indicative of hereditary or sporadic cancer. Further, undue experimentation would be required to determine how expression products other than those listed in Table 17 would be indicative of microsatellite stability/instability. Such experimentation would be inventive.

The level of unpredictability for using the presence or particular expression pattern of a particular molecule (or molecules) to detect any disease state is quite high. The state of the prior art dictates that one of skill in the art would not predict that a particular expression pattern of a particular molecule is indicative of a particular diseased state without a demonstration that said particular diseased state correlates with said particular expression pattern of said particular molecule. For example, Tockman et al (Cancer Res., 1992, 52:2711s-2718s) teach considerations necessary in bringing a cancer biomarker (intermediate end

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point marker) to successful application. Absent evidence demonstrating a particular expression pattern of particular molecules correlating with a particular diseased state, one of skill in the art would not predict said particular expression pattern of said particular molecules correlates with said particular diseased state without undue experimentation. Experimentation to identify such a correlation would in itself be inventive.

It is further noted that evidence abounds in which protein levels do not correlate with alterations in mRNA levels. There are many steps in the pathway leading from DNA to protein, and all of them can, in principle, be regulated. For example, Alberts *et al.* (Molecular Biology of the Cell, 3rd edition, 1994, page 465) illustrate post-transcriptional regulation of ferritin wherein the translation of ferritin mRNA into ferritin polypeptide is blocked during periods of iron starvation. Likewise, if excess iron is available, the transferrin receptor mRNA is degraded and no transferrin receptor polypeptide is translated. Further, Greenbaum *et al.* (Genome Biology, 2003, Vol. 4, Issue 9, pages 117.1-117.8) cautions against assuming that mRNA levels are generally correlative of protein levels. The reference teaches (page 117.3, 2nd column) that primarily because of a limited ability to measure protein abundances, researchers have tried to find correlations between mRNA and the limited protein expression data, in the hope that they could determine protein abundance levels from the more copious and technically easier mRNA experiments. To date, however, there have been only a handful of efforts to find correlations between mRNA and protein expression levels, most notably in human cancers and yeast cells. And, for the most part, they have

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reported only minimal and/or limited correlations. The reference further teaches (page 117.4, 2nd column) that there are presumably at least three reasons for the poor correlations generally reported in the literature between the level of mRNA and the level of protein, and these may not be mutually exclusive. First, there are many complicated and varied post-transcriptional mechanisms involved in turning mRNA into protein that are not yet sufficiently well defined to be able to compute protein concentrations from mRNA; second, proteins may differ substantially in their *in vivo* half lives; and/or third, there is a significant amount of error and noise in both protein and mRNA experiments that limit our ability to get a clear picture. The reference further notes (page 117.6, page 2nd column) that to be fully able to understand the relationship between mRNA and protein abundances, the dynamic processes involved in protein synthesis and degradation have to be better understood. Thus, due to the multitude of homeostatic factors affecting transcription and translation, protein levels do not predictably correlate with levels of mRNA (and vice-versa).

One cannot extrapolate the teachings of the specification to the scope of the claims because the claims are broadly drawn to methods wherein a colon cancer patient with just any amount of just any polypeptide or polynucleotide expressed by a gene in Table 13 in just any sample from the colon cancer patient is classified, using just any determination(s) to classify, as having a hereditary cancer, a sporadic cancer, a microsatellite stable cancer, and/or a microsatellite instable cancer, and Applicant has not enabled said methods because it has not been shown that just any determinations used to classify would predictably

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classify a colon cancer patient as having a hereditary cancer, a sporadic cancer, a microsatellite stable cancer, and/or a microsatellite instable cancer with any predictability of success.

In view of the teachings above and the lack of guidance, workable examples and or exemplification in the specification, it would require undue experimentation by one of skill in the art to determine with any predictability, that the method would function as claimed.

In the Reply of 8/2/11, Applicant argues the claims are enabled because the specification discloses that genes in Table 13 have been definitively established as classifiers of sporadic vs. hereditary colon cancer. Applicant further states that the claims do not specify that certain genes are up-regulated or down-regulated in a pattern to determine hereditary or sporadic nature of cancer and indicates that the claims encompass methods wherein markers that are disclosed as being indicative of hereditary disease when downregulated (such as PIWIL1) could be indicative of sporadic disease when downregulated. Applicant further indicates that amendments to claim 68 would prevent the markers from being polypeptide markers.

The amendments to the claims and the arguments found in the Reply of 8/2/11 have been carefully considered, but are not deemed persuasive. In regards to the argument that the claims are enabled because the specification discloses that genes in Table 13 have been definitively established as classifiers of sporadic vs. hereditary colon cancer, the specification does not disclose in what way all genes of Table 13 classify sporadic vs. hereditary colon cancer.

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Undue experimentation would be required to determine how expression products other than MLH1 polynucleotides and PIWIL1 polypeptides would be indicative of hereditary or sporadic cancer.

In regards to the argument that that the claims do not specify that certain genes are up-regulated or down-regulated in a pattern to determine hereditary or sporadic nature of cancer and indicates that the claims encompass methods wherein markers that are disclosed as being indicative of hereditary disease when downregulated (such as PIWIL1 polynucleotide) could be indicative of sporadic disease when downregulated, the examiner agrees that the claims broadly encompass methods wherein markers that are disclosed as being indicative of hereditary disease when downregulated (such as PIWIL1) could be interpreted to be indicative of sporadic disease when downregulated. However, the claims in view of the specification do not recite which results are indicative of sporadic disease and which results are indicative of hereditary disease. The claims encompass methods wherein just any result is equally indicative of sporadic disease and hereditary disease. Such a method would not predictably function as claimed because the specification discloses PIWIL1 polynucleotides are downregulated with hereditary disease and one of skill in the art would not predict that the same result would be equally indicative of sporadic disease. Absent evidence demonstrating a particular expression pattern of particular molecules correlating with a particular diseased state, one of skill in the art would not predict said particular expression pattern of said particular molecules correlates with said particular diseased state without undue experimentation.

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In regards to the indications that amendments to claim 68 would prevent the markers from being polypeptide markers, claim 68 recites that the markers are "gene expression products...expressed by a gene in Table 13". Polypeptides are, clearly, gene expression products expressed by genes of Table 13 (also see withdrawn claim 91).

Claim 94 is rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of determining/classifying microsatellite status of a colon cancer wherein levels of ATP9a polynucleotide, FLJ20618 polynucleotide, SET polynucleotide, and PRKCBP1 polynucleotide are elevated in microsatellite stable tumors as compared to microsatellite instable tumors and levels of HNRPL polynucleotide, MTA1L1 polynucleotide, SFRS6 polynucleotide, CXCL10 polynucleotide, and PRKCBP1 polynucleotide are elevated in microsatellite instable tumors as compared to microsatellite stable tumors, **the specification does not reasonably provide enablement for** methods wherein just any pattern of polynucleotide and polypeptide levels expressed by genes of instant Table 17 in just any type of patient sample from an individual with colon cancer is indicative of just any microsatellite status. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to practice the invention commensurate in scope with these claims.

Factors to be considered in determining whether undue experimentation is required are summarized in *Ex parte* Forman, 230 USPQ 546 (BPAI 1986). They

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include the nature of the invention, the state of the prior art, the relative skill of those in the art, the amount of direction or guidance disclosed in the specification, the presence or absence of working examples, the predictability or unpredictability of the art, the breadth of the claims, and the quantity of experimentation which would be required in order to practice the invention as claimed.

The instant claims are drawn to methods wherein just any pattern of polynucleotide and polypeptide levels expressed by genes of instant Table 17 in just any type of patient sample from an individual with colon cancer is indicative of just any microsatellite status. This includes methods of determining microsatellite status based on polypeptide levels. This includes contradictory methods wherein polynucleotides that are disclosed as being elevated in microsatellite stable tumors are predictably equally indicative of microsatellite stable and unstable tumors when elevated.

This invention is in a class of invention which the CAFC has characterized as "the unpredictable arts such as chemistry and biology". *Mycogen Plant Sci., Inc. v. Monsanto Co.*, 243 F.3d 1316, 1330 (Fed. Cir. 2001).

The specification demonstrates a method of determining/classifying microsatellite status of a colon cancer wherein levels of ATP9a polynucleotide, FLJ20618 polynucleotide, SET polynucleotide, and PRKCBP1 polynucleotide are elevated in microsatellite stable tumors as compared to microsatellite unstable tumors and levels of HNRPL polynucleotide, MTA1L1 polynucleotide, SFRS6 polynucleotide, CXCL10 polynucleotide, and PRKCBP1 polynucleotide are

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elevated in microsatellite instable tumors as compared to microsatellite stable tumors (see Table 17, in particular). The specification does not demonstrate the method functions when using polypeptide levels as claimed. Further, the specification does not demonstrate contradictory methods wherein polynucleotides that are disclosed as being elevated in microsatellite stable tumors are predictably equally indicative of microsatellite stable and instable tumors when elevated. Further, possible limitations disclosed in the specification are not read into the claims.

The level of unpredictability for using the presence or particular expression pattern of a particular molecule (or molecules) to detect any disease state is quite high. The state of the prior art dictates that one of skill in the art would not predict that a particular expression pattern of a particular molecule is indicative of a particular diseased state without a demonstration that said particular diseased state correlates with said particular expression pattern of said particular molecule. For example, Tockman et al (Cancer Res., 1992, 52:2711s-2718s) teach considerations necessary in bringing a cancer biomarker (intermediate end point marker) to successful application. Absent evidence demonstrating a particular expression pattern of particular molecules correlating with a particular diseased state, one of skill in the art would not predict said particular expression pattern of said particular molecules correlates with said particular diseased state without undue experimentation. Experimentation to identify such a correlation would in itself be inventive.

It is further noted that evidence abounds in which protein levels do not correlate with alterations in mRNA levels. There are many steps in the pathway leading from DNA to protein, and all of them can, in principle, be regulated. For example, Alberts *et al.* (Molecular Biology of the Cell, 3rd edition, 1994, page 465) illustrate post-transcriptional regulation of ferritin wherein the translation of ferritin mRNA into ferritin polypeptide is blocked during periods of iron starvation. Likewise, if excess iron is available, the transferrin receptor mRNA is degraded and no transferrin receptor polypeptide is translated. Further, Greenbaum *et al.* (Genome Biology, 2003, Vol. 4, Issue 9, pages 117.1-117.8) cautions against assuming that mRNA levels are generally correlative of protein levels. The reference teaches (page 117.3, 2nd column) that primarily because of a limited ability to measure protein abundances, researchers have tried to find correlations between mRNA and the limited protein expression data, in the hope that they could determine protein abundance levels from the more copious and technically easier mRNA experiments. To date, however, there have been only a handful of efforts to find correlations between mRNA and protein expression levels, most notably in human cancers and yeast cells. And, for the most part, they have reported only minimal and/or limited correlations. The reference further teaches (page 117.4, 2nd column) that there are presumably at least three reasons for the poor correlations generally reported in the literature between the level of mRNA and the level of protein, and these may not be mutually exclusive. First, there are many complicated and varied post-transcriptional mechanisms involved in turning mRNA into protein that are not yet sufficiently well defined to be able to compute

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protein concentrations from mRNA; second, proteins may differ substantially in their *in vivo* half lives; and/or third, there is a significant amount of error and noise in both protein and mRNA experiments that limit our ability to get a clear picture. The reference further notes (page 117.6, page 2nd column) that to be fully able to understand the relationship between mRNA and protein abundances, the dynamic processes involved in protein synthesis and degradation have to be better understood. Thus, due to the multitude of homeostatic factors affecting transcription and translation, protein levels do not predictably correlate with levels of mRNA (and vice-versa).

One cannot extrapolate the teachings of the specification to the scope of the claims because the claims are broadly drawn to methods wherein just any pattern of polynucleotide and polypeptide levels expressed by genes of instant Table 17 in just any type of patient sample from an individual with colon cancer is indicative of just any microsatellite status, and Applicant has not enabled said methods because it has not been shown that just any pattern of polynucleotide and polypeptide levels expressed by genes of instant Table 17 in just any type of patient sample from an individual with colon cancer is indicative of just any microsatellite status.

In view of the teachings above and the lack of guidance, workable examples and or exemplification in the specification, it would require undue experimentation by one of skill in the art to determine with any predictability, that the method would function as claimed.

In the Reply of 8/2/11, Applicant the specification discloses enabling results in Table 17.

The amendments to the claims and the arguments found in the Reply of 8/2/11 have been carefully considered, but are not deemed persuasive. In regards to the argument that the specification discloses enabling results in Table 17, Table 17 demonstrates a method of determining/classifying microsatellite status of a colon cancer wherein levels of ATP9a polynucleotide, FLJ20618 polynucleotide, SET polynucleotide, and PRKCBP1 polynucleotide are elevated in microsatellite stable tumors as compared to microsatellite instable tumors and levels of HNRPL polynucleotide, MTA1L1 polynucleotide, SFRS6 polynucleotide, CXCL10 polynucleotide, and PRKCBP1 polynucleotide are elevated in microsatellite instable tumors as compared to microsatellite stable tumors. However, Table 17 does not demonstrate broadly-claimed methods wherein just any pattern of polynucleotide and polypeptide levels expressed by genes of instant Table 17 in just any type of patient sample from an individual with colon cancer is indicative of just any microsatellite status.

Summary

No claim is allowed.

Conclusion

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Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to SEAN AEDER whose telephone number is (571)272-8787. The examiner can normally be reached on M-F: 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Misook Yu can be reached on 571-272-0839. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

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Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Sean E Aeder/
Primary Examiner, Art Unit 1642